REPORT OF ACTIVITIES

of the

PARASITOLOGY PROJECT

of the

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PRELIMINARY REPORT OF STUDIES ON TYPHUS, LEPTOSPIROSIS AND TRICHINOSIS IN HONOLULU

by

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CONTENTS

A. INTRODUCTION

Rat-borne diseases are subjects of special interest in Hawaii because of the widespread distribution of rodents and because in recent years several human diseases believed to be of rodent origin have been uncovered. Interest in rat-borne diseases in Hawaii was aroused in the latter part of 1899 when bubonic plague made its appearance in Honolulu. In more recent time (1933) with the first recognition of typhus fever in man locally and the finding of typhus infection in local rats by Dr. L. F. Badger (cited by Doolittle, 13), the public health importance of rodents was again realized. According to the report by Dr. S. E. Doolittle (13) cases of human typhus fever in the Territory had undoubtedly occurred prior to that time. The disease was unrecognized largely because the biological and clinical distinction between epidemic typhus (man-to-louse and louse-to-man transmission) and endemic typhus (rat-to-flea and flea-to-man transmission) was not clearly known until the work of Dyer, Rumreich and Badger in 1931 (15). Following the recognition of human clinical trichinosis by Dr. W. N. Bergin in 1936, and a short time later the demonstration by the writer (1, 2, 3) of trichinae larvae in animals (rats, mongooses, wild and domesticated hogs), rats were believed to play an important part as reservoir hosts and in the dissemination of the porcine infection. After reviewing the importance of rats in the local transmission of plague, rat-bite fever, endemic typhus and trichinosis, Dr. E. A. Fennel (19) reported, "As far as I know, no proven case of infectious jaundice which is carried by rats, has been reported in Hawaii, but all idiopathic jaundice cases must be looked on with suspicion." A few months after the publication of the above statement, the writer (2) isolated, through guinea pig inoculations, the causative agent of infectious jaundice from a plantation worker suffering from acute jaundice. This patient had been referred by the attending physician, Dr. Thomas Keay, of Pepeekeo Hospital on the Island of Hawaii. Subsequently, leptospiral infection was demonstrated in rats trapped on the plantation where the patient had worked.

Of typhus and leptospirosis, little was known regarding their prevalence among the rodent population. The mode of transmission of these diseases to man, particularly that of typhus fever, were and still are not clearly understood. In order to elucidate some of these problems, which have considerable bearing on the application of proper control measures, funds were appropriated by the Public Health Committee, Chamber of Commerce of Honolulu, to establish the Parasitology Project. The report which follows presents investigations carried out during the year of 1941 and also summarizes certain facts related to these studies which are of local interest. In these studies considerable credit is due Miss Virginia Breaks, Research Assistant, who has aided the writer in carrying out all phases of this investigation.

Unless interrupted by the present world crisis, all phases of these studies will be continued throughout the year 1942.

B. STUDIES ON TYPHUS

I. Local Importance of Typhus Fever in Man

Up to the present time human typhus fever in the Hawaiian Islands has been of major importance on the Island of Oahu, particularly in Honolulu. A few cases however have been reported in recent years from Hawaii, Maui, Kauai and Molokai.

From July 1, 1933 to June 30, 1934, the first year that the disease was recognized here, 5 cases were reported in Honolulu. From July, 1934 through June, 1941, 248 cases have been reported, varying from 18 to 57 each year and averaging 35.4 a year. During the last six months of 1941, 47 cases were reported in Honolulu, this number represents about twice as many cases as were reported from any similar period heretofore. There is a possibility that a certain amount of this apparent increase is due to wider reporting of cases.

A summary of the distribution of known typhus cases reported to the Territorial Board of Health, is given in table 1. Table 2 shows the occurrence by month.

TABLE I

REPORTED CASES OF ENDEMIC TYPHUS IN THE HAWAIIAN ISLANDS

Period (Fiscal year beginning July 1 and ending June 30)	Honolulu City(Oahu)	Rural Oahu	Hawaii	Maui	Kauai	Molokai	Total cases
1933 - 1934	5	1	0	0	0	0	6
1934 - 1935	18	3	0	0	0	0	21
1935 - 1936	22	2	0	7	0	0	31
1936 - 1937	49	5	6	0	0	0	60
1937 - 1938	28	13	1	0	1	2	45
1938 - 1939	37	4	8	3	3	2	57
1939 - 1940	57	2	11	1	3	3	77
1940 - 1941	37	7	7	9	2	2	64
July - Dec. 1941	47	4	1	1	2	0	55
TOTAL	300	41	34	21	11	9	416

TABLE II

VARIATIONS IN THE MONTHLY INCIDENCE OF HUMAN TYPHUS FEVER IN HONOLULU DURING A 5-YEAR PERIOD (1936-1941)

	1937	1938	1939	1940	1941	Total	Average
Jan.	3	0	1	2	1	7	1.4
Feb.	0	1	1	4	0	6	2.4
Mar.	2	2	1	2	1	8	1.6
Apr.	0	1	0	5	7	13	2.6
May	1	2	1	2	3	9	1.8
June	1	5	1	4	4	15	3.0
July	3	3	3	4	1	14	2.8
Aug.	1	4	2	6	3	16	3.2
Sept.	4	5	13	4	14	40	8.0
Oct.	2	10	7	2	15	36	7.2
Nov.	3	7	6	4	7	27	5.4
Dec.	4	6	7	1	7	25	5.0

II. Incidence of Typhus Infection in Rats from Various Districts of Honolulu.

(a) Purpose of the investigation

"Knowledge of the strength of the enemy is as necessary in the control of human parasites as it is in a war between nations" (21). This investigation was made in order to obtain information regarding the extent to which typhus virus has already invaded the murine territory in Honolulu. It was also believed that such data would be useful in (a) demonstrating the seriousness of the problem, (b) determining relationships between the incidence of human and murine infections, and (c) pointing out areas where rat control might be more intensively carried out.

This survey is at present incomplete, but additional data will be obtained during the coming year.

(b) Methods of Investigation

In this survey, 100 rats were trapped from each of the several districts of Honolulu. The rats were trapped alive in cage traps and after being

killed by gas in a closed chamber were immersed in kerosene for the purpose of killing any fleas present. The skull of each animal was then opened aseptically and about one-fourth to one-half of the brain removed. The brains of two to five rats were pooled and emulsified in about 20 cc. of sterile physiological saline, and 3 cc. of this emulsion were inoculated intraperitoneally into a grown male guinea pig weighing from 400 to 600 grams. Following inoculation daily rectal temperatures of each guinea pig were recorded for a period of 15 to 20 days. The animals which developed clinical typhus (fever and scrotal swelling) were, after 15 days or longer following recovery (i.e. absence of fever and scrotal involvement), tested for typhus immunity by inoculating them with a known strain of endemic typhus. This strain (Wilmington) was secured through Drs. R. E. Dyer and N. A. Topping of the National Institute of Health, U. S. Public Health Service, Washington, D. C. and was maintained in the laboratory in guinea pigs. In this study all the guinea pigs which developed clinical typhus as a result of the rat-brain inoculation showed complete immunity when inoculated with the Wilmington typhus strain.

The guinea pigs which did not show clinical typhus during the first 30 days following the inoculation with the rat-brains were inoculated with the known strain of typhus to determine the possibility of their having passed through a stage of sub-clinical typhus (i.e. having developed typhus without showing fever and scrotal involvement). In the course of this study, there were a few guinea pigs which had developed sub-clinical typhus as evidenced by complete protection against the Wilmington typhus strain. In all these experiments fresh control male guinea pigs were also inoculated in order to ascertain the pathogenicity of the strain at the time of the passage.

Up to the present time rats have been trapped within four districts of Honolulu, as follows:

1. Kaimuki district, within the area approximately bounded by 20th Avenue, Kilauea Avenue, Makapuu Avenue, Monsarrat Avenue, Kanaina Avenue, Kapahulu Avenue, Fourth Avenue, Palolo Avenue, Pukele Avenue, Hardesty Street and Sierra Drive.

2. Waikiki district, within the area bounded by Kalakaua Avenue, Kalia and Ena Roads, Ala Wai Canal and Kapahulu Avenue.

3. Manoa- Bingham district, within the area approximately bounded by Manoa Road, Lowrey Street, East Manoa Road, University Avenue, King Street, Hausten Street, Algaroba and Alexander Streets.

4. Downtown or Business district, within the area bounded by Ala Moana, Nuuanu Avenue, Iolani Avenue and Ward Avenue.

(c) Experimental data

A summary of the guinea pigs which were inoculated with emulsion of brains from rats trapped in four districts of Honolulu is given in table 3.

TABLE III

SUMMARY OF TYPHUS INFECTION IN RATS
FROM FOUR DISTRICTS OF HONOLULU

District surveyed	Number of rats examined	Number of guinea pigs inoculated with pooled brain emulsion of rats	Number of guinea pigs developing clinical typhus	Number of guinea pigs showing subclinical typhus	Minimum number of rats harboring typhus virus in brain as revealed from number of positive guinea pig reactions	
Kaimuki	100	40	11	3	14	(14.0%)
Waikiki	100	42	2	1	3	(3.0%)
Manoa-Bingham	100	42	6	3	9	(9.0%)
Downtown	100	43	21	0	21	(21.0%)
TOTAL	400	167	40	7	47	(11.7%)

As explained above each guinea pig received an inoculation of a pooled brain emulsion of two to five rats. If the guinea pig developed typhus, it could not be determined whether only one or more than one of the rat brains received by the animal actually harbored typhus virus. Therefore the number of rats reported as infected represents only the minimum; the rate of infection among the rats was probably slightly higher than indicated.

The finding of infected rats in any locality does not necessarily imply that these animals were active typhus carriers at the time of examination. In animals which acquire typhus infection the virus circulates in the blood possibly for not more than a few weeks; it is during this period that ectoparasites found on the animal acquire the infection. Fleas have been found to maintain the infection after 52 days (14) and 100 days (6). The virus in the mammalian host, following its presence in the blood, localizes in several body tissues including the brain. The virus can be recovered in the brains of rats for a period of about a year following infection; laboratory experiments conducted by Philip and Parker (29) showed that typhus virus was recovered from the brain of white rats 370 days but not 463 days after infection.

As revealed in the present limited survey, differences exist in degree of infection in the four districts examined. These may be related to differences in the prevalence of rats in the districts. Localities with an abundance of old and improperly rat-proofed dwellings, empty and unkept lots with many kiawe trees, stone walls, and other conditions which offer food and shelter, will undoubtedly maintain a larger rat population than where the opposite is true. It is interesting in this connection that few cases

of murine infection were noted in the district of Waikiki which appears to possess less rat harborages than other areas surveyed. The possible cause for the high prevalence of typhus infection among rats in the downtown area, in addition to the high incidence of rats, may be the closer intermingling of these animals because of more overcrowded conditions.

III. Possible Source of Human Infection with Typhus Fever

(a) Opinions suggested by various investigators

Up to the present time rats, and to some extent mice, have been regarded as the original source of human infection. The spread of the disease from rat to rat is believed to be brought about through contamination with feces of infected fleas, the virus gaining entrance into the body through the broken skin (14), contact with the mucous membranes of the mouth, nose, and eyes (8), and by the digestive route as a result of cannibalism among rats or the ingestion of infected fleas (32). It is questionable whether typhus can be brought about through the bite of an infected flea; Ceder and collaborators (10) were unable to transmit the disease by allowing infected fleas to feed on guinea pigs.

Since fleas remain with the rat host for a considerable time, the hairs and skin of these animals as well as the surroundings become contaminated with the feces of infected fleas; the virus in the feces of fleas can remain alive for at least 651 days and a very small amount (0.01 mg.) is sufficient to produce an infection (7). With these conditions, it is readily understood how inter-murine infection is brought about.

When it comes to the question of human infection, a satisfactory explanation is not available. As a result of an experiment in which guinea pigs acquired typhus infection by scratching the skin with feces of infected fleas, Dyer and collaborators (14) suggested that a probable mechanism by which endemic typhus is transmitted is through rubbing of infected flea feces into wounds made by the biting of the flea or by scratching. It was also suggested by Blanc and collaborators (8) that human infection through the mucous membrane, as a result of contact with feces of infected fleas, appears to be the most usual method of transfer in nature.

The suggestion that human infection is acquired as a result of rubbing infected flea feces in the wound made by the bite of the flea or by scratching, although possible, does not seem very probable. Fleas as a rule remain attached to the skin of humans only a short time sufficient to take a blood meal, and it is improbable that they will deposit feces at the interim of feeding. This supposition is substantiated by the observations of Eskey (17, pg. 31) who, in connection with the supposed transmission of plague to man by the rubbing of the feces of fleas on the skin, reports: "It has been stated that plague infection may be induced by rubbing infected feces, which has been deposited by fleas when feeding, into the skin, but this means of infection would be extremely rare if it ever occurred, because itching does not usually follow bites, and in only two instances out of several hundred feedings on human blood were feces deposited on the skin."

The suggestion of human infection through the mucous membranes appears to offer a more satisfactory explanation, although this, too, implies a close association of man with the infected agent. As is already known, rats and mice, through their fleas are the known infective agents. The close association of man with these rodents, although true in a larger percent (84.6%) of 39 cases investigated in Honolulu (reported on later in this paper), does not seem true in all cases. It is of interest in this connection to quote from Maxcy's (26) report on an epidemiological study of endemic typhus in the southeastern United States. "The occupational analysis also brings out the fact that the disease attacked, for the most part, persons earning a reasonably good livelihood. There is a notable absence of cases among unskilled laborers and unemployed males.....the disease did not select the poor and uncleanly.....There is no particular association of the disease with cheap boarding and rooming houses."

Based on the question of the transfer of typhus from rat to man, the writer thought it desirable to investigate the possibility of typhus transfer from the rat to cats and dogs, and from these animals to man. This opinion originated as a result of reports that cats (23, 24) and dogs (12) and their fleas were susceptible to typhus infection. In addition, certain European investigators (11, 24) reported finding typhus virus in the brains of cats, and fleas from these cats, in houses where human typhus had occurred.

The information which follows summarizes the experiments and results obtained to date regarding the susceptibility and transmission of typhus by cats and dogs in nature.

(b) Experiments to determine susceptibility of cats, dogs, and their fleas to typhus virus.

Experiment I. A young cat was infected intraperitoneally with the testicular washings of an experimentally typhus-infected guinea pig. At the same time, a large number of laboratory-raised cat fleas (Ctenocephalides felis) were placed on the cat. Ten days later the cat was killed by drowning. A small amount of the brain of the cat was removed aseptically and, after being emulsified in saline solution, inoculated into a male guinea pig.

At the same time the cat was killed, about 100 fleas were recovered. Fifteen of these fleas which remained alive were placed and kept in a small test tube overnight. The following morning the fleas were removed from the test tube. The feces of the fleas, adhering to the walls of the test tube, were taken up in saline solution and inoculated into a male guinea pig. The remainder of the fleas removed from the cat, after being properly washed and macerated in saline solution, were inoculated into a male guinea pig. A few days following inoculation the guinea pigs developed clinical typhus and were subsequently found immune when inoculated with the Wilmington strain of typhus.

The above experiments demonstrated that cats and dogs are susceptible to typhus and are able, at least in the case of cats, to pass on the infection to the fleas which they harbor. These findings also confirm the reports of other investigators (12, 23, 24) regarding the susceptibility of these animals to typhus.

(c) Observations to determine possible occurrence of typhus virus among stray cats and dogs.

Examination of cats. This investigation was conducted because it was believed possible for cats and dogs to acquire typhus infection as a result of their close association with rats, or by eating infected rats and their fleas. During the course of this study 30 guinea pigs were inoculated with the pooled brains of 109 cats. Of these cats, 102 were fullgrown and 7 were young. Seven of the grown cats were secured from residences where human typhus fever cases had recently occurred. Of the guinea pigs inoculated, none developed clinical typhus nor were they found immune when inoculated with the Wilmington typhus strain. In addition, the fleas which were recovered from the 7 cats from human typhus residences failed to produce typhus when inoculated into guinea pigs.

The above results appear to indicate that under natural conditions cats are not directly responsible for the dissemination of typhus. There is a likelihood however that the negative results secured were due to the fact that most of the animals examined were full grown. It may be that in areas where murine typhus occurs, cats acquire the infection while young, and, since the virus loses its virulence in the brain of the cat within a relatively short time, this may explain the reason for our negative findings. (Reference regarding the loss-virulence of exanthematous typhus in the brain of the cat after 69 days is found in abstract published in Revue de Medicine Veterinaire, v. 91, Apr. 1939; also in Jour. Am. Vet. Med. Assoc., v. 96 (756), March, 1940, p. 364). The above experiments based on the use of brains from grown cats should be considered as inconclusive and, therefore, additional experiments should be conducted using the brains of young cats.

Examination of dogs. Up to the present time 11 guinea pigs have been inoculated with pooled brain emulsions of 26 dogs. In no cases did these animals show evidence of typhus infection. In addition to the brain inoculations, the sera of 46 additional dogs were subjected to a Weil-Felix test. Of these, 26 were negative, two showed agglutination in dilutions of 1:10, six in 1:20, five in 1:40, two in 1:80, and one in 1:160. Because these sera agglutinated only at low dilutions, the results do not appear to be highly significant.

(d) Local rat fleas as known carriers of murine typhus

According to reports by Eskey (17) there are five species of fleas known to infest rats on the island of Oahu; namely: Xenopsylla cheopis, Echidnophaga gallinacea, Ctenocephalides felis, Pulex irritans and Xenopsylla hawaiiensis. In addition to these, Leptosyllus segnis and Nasopsyllus fasciatus have been found on rats on the islands of Hawaii and Maui. Also according to Eskey, of the rats found infested with fleas on Oahu, X. cheopis occurred in 43 percent, E. gallinacea in 11 percent, C. felis in 5 percent, X. hawaiiensis in 2 percent, and Pulex irritans rarely. In reference to the abundance or percentage of all these fleas recovered from the rats, about 55 percent were E. gallinacea; about 44 percent, X. cheopis; about 2 percent, C. felis, and about 2 percent, X. hawaiiensis.

Of the species of fleas found locally, the following were, previous to this investigation, reported to be suitable carriers of typhus: X. cheopis (15), C. felis (28), P. irritans (28), L. segnis (28), and N. fasciatus (16).

Since the sticktight or chicken flea, E. gallinacea, is a common flea of local rats, and since it is also found to be common to local dogs and cats, an experiment was conducted to determine the susceptibility of these fleas to endemic typhus. Several of these fleas were allowed to attach to a rat which had recently been inoculated with the Wilmington strain of endemic typhus. Thirteen days later an emulsion made from the feces recovered from the fleas, as well as an emulsion of the macerated bodies of these fleas, produced clinical endemic typhus when inoculated into male guinea pigs. The strain from the fleas was thereafter successfully passed in four generations of guinea pigs and also into a rabbit. The serum of the latter demonstrated agglutinins for B. proteus OX_{19} following the infection. The results of these findings have been submitted for publication (5).

Following the above observation, Brigham (9) reported recovering two strains of endemic typhus fever virus in sticktight fleas trapped from wild rats in Georgia. These findings confirmed the observations of the writer as well as pointing out the importance of these fleas under natural conditions.

In connection with flea-infection among rats, two species, C. felis and E. gallinacea, also commonly infest local dogs and cats. During the present investigations, we observed the margins of the ears of many dogs and cats covered with hundreds of sticktight fleas, E. gallinacea; this flea has also been found common to local poultry.

It is not uncommon to find large numbers of fleas in basements of houses and other areas which are frequented by stray cats. Undoubtedly there must be an exchange of fleas among these animals; stray or ill-kept dogs and cats, and poultry may assist in the maintenance of flea populations among rats.

IV. Type of Typhus Fever Occurring in Local Humans

Cases of human typhus fever were first recognized and reported by Fennel in 1933 (18). The diagnosis was based on clinical findings, positive Weil-Felix reactions and protection tests carried out by the U. S. Public Health Service in guinea pigs with the serum of one of the suspected cases.

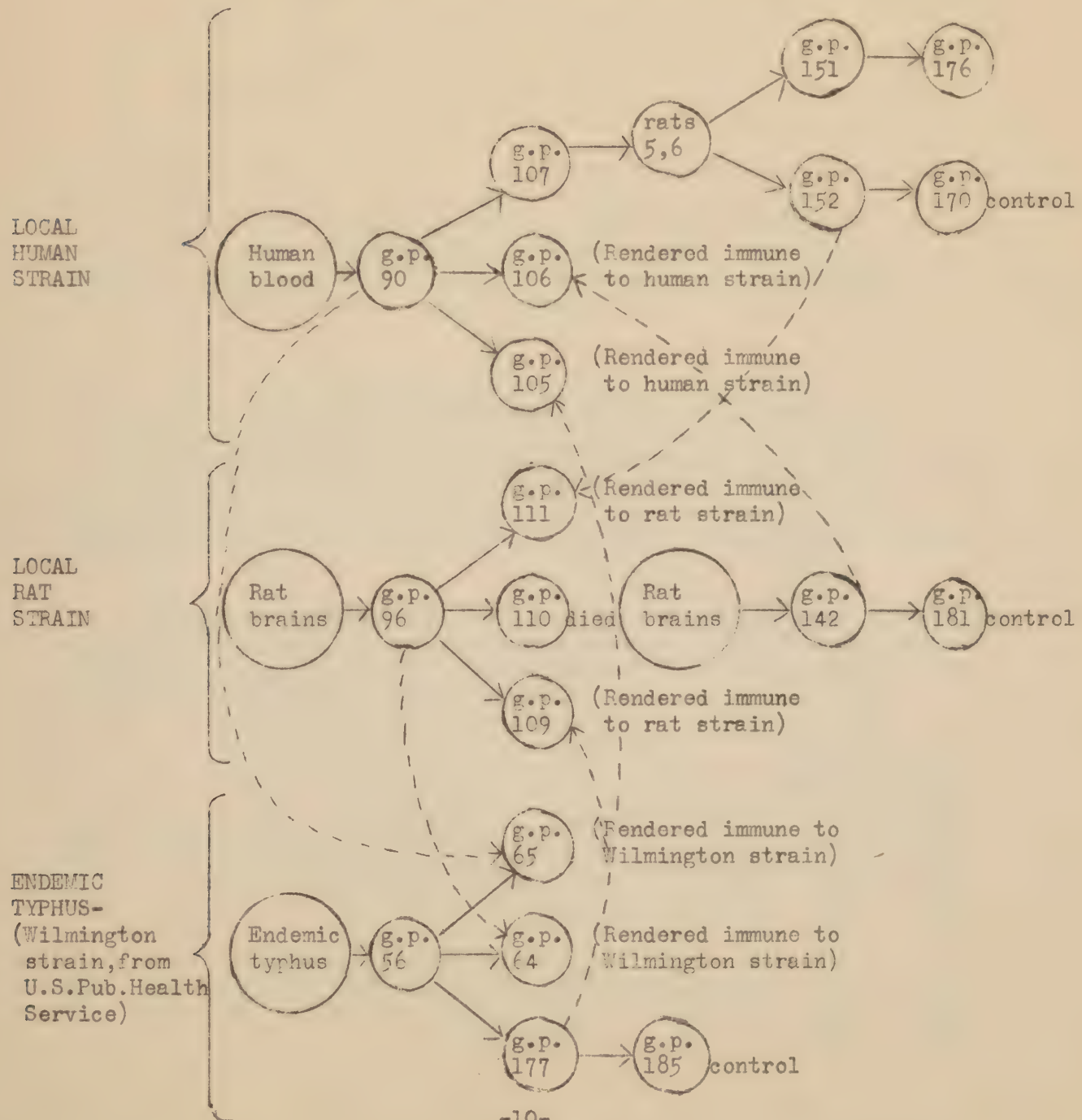
A short time later Badger (reported by Doolittle, 13) detected the presence of endemic typhus in the brain of local rats as a result of guinea-pig inoculations; infected guinea pigs were submitted to the National Institute of Health, Washington, D. C. and experiments conducted there showed that the virus was clinically and immunologically identical to their Wilmington strain of endemic typhus. These findings led to the assumption that the form of typhus found locally in humans was of murine origin, hence endemic.

In the course of this study opportunity arose to add more evidence regarding the type of local human typhus. Cross immunity tests were carried out between the following three strains of typhuses: A strain of local human typhus (recovered from a suspected case and obtained through the cooperation

of Dr. J. W. McClellan); a strain of murine typhus (recovered from local infected rats) and a strain of endemic typhus (Wilmington strain) obtained from the U. S. Public Health Service.

The cross immunity tests mentioned above which were conducted in male guinea pigs (g.p.) are illustrated below.

FIG. I. CROSS-IMMUNITY TESTS AMONG LOCAL HUMAN AND RAT STRAINS AND WILMINGTON STRAIN OF TYPHUS VIRUSES.



In the above experiments typhus was not transmitted from:

(a) The human strain, to a guinea pig which had been immunized to the rat strain (from g.p. 152 into g.p. 111, already immune to the rat strain), and a guinea pig immunized to the Wilmington strain (g.p. 90 into immune g.p. 65).

(b) The rat strain, to a guinea pig immune to the human strain (g.p. 142 into immune g.p. 106), and to a guinea pig immunized for the Wilmington strain (g.p. 96 into immune g.p. 64). In the plan of this experiment g.p. 110 was intended to be used in the continuation of passage of this strain. Since this animal died, another rat strain was substituted.

(c) The Wilmington strain, to a guinea pig immune to the human strain (g.p. 177 into immune g.p. 105) and to a guinea pig immune to the rat strain (g.p. 177 into immune g.p. 109).

Of the passages conducted in testing for cross immunity, all the control animals developed typical fever and scrotal swelling. The fact that all the guinea pigs inoculated with the human strain developed fever and scrotal involvement and subsequently showed complete protection when inoculated with both the rat and the Wilmington strains of typhus, is sufficient evidence that the human strain was of endemic typhus.

V. Environmental Conditions Associated with Cases of Human Typhus.

This study was conducted for the purpose of ascertaining any correlation between the occurrence of typhus with that of the patient's environmental conditions. Data have thus far been obtained from 39 patients (26 males, 13 females) who had recently recovered from typhus infection.

The group from which data have been secured is too small to establish definite conclusions, but the available evidence points out the probable validity of certain tentative assumptions which are made. The 39 individuals interviewed reported as follows:

		Number reporting	Percent
(a)	Presence of rats at home only.....	21	53.8
(b)	" " " both at home and at place of work.	9	23.0
(c)	" " " at place of work only.....	3	7.6
	Total a, b, c.....	23	84.6
(d)	" " mice at home only.....	15	38.4
(e)	" " " both at home and at place of work	8	20.5
(f)	" " " at work only.....	1	2.5
	Total d, e, f.....	24	61.5

		Number Reporting	Percent
(g)	Handled rats	4	10.2
(h)	Were bitten by fleas	17	43.5
(i)	Had seen fleas at home	13	33.3
(j)	Had kept cats at home only	4	10.2
(k)	" " cats and dogs at home	3	7.6
(l)	" " dogs at home only	5	12.8
	Total j, k, l	12	30.7
(m)	Raised poultry	12	30.7
(n)	" " by neighbor	3	7.6
	Total m, n	15	38.4

The above data clearly demonstrate a close relationship between the incidence of rats (84.6%) and human cases of typhus fever. It also points out that the infection most likely took place at home rather than at the place of work.

That rats played an important part in the transmission of typhus was also shown by the finding of typhus infection in rats trapped in residences where human typhus fever occurred. Of rats trapped in 17 residences, murine infection was found in eight or 47.0 percent of the cases.

So far as the individuals who handled rats are concerned, the infection probably followed as a result of contact with the typhus virus present on the body of the rat. One of these individuals was a radio technician who, several days before illness, took a rat out of a radio box. Since the rat was reported as having been dead for sometime, there is a possibility that fleas were absent. This finding points out that, in this case, infection probably took place through contact, i.e. contamination of the fingers and subsequent touching of some exposed mucous membrane such as of the mouth, nasal passages or eyes.

The fact that about one-half of these persons (43.5%) reported as having been bitten by fleas, is indicative that they had been in places where fleas were present. Little correlation can be drawn from this item since flea-bites are not uncommon in this locality and many of the fleas are probably from uninfected canine or feline sources.

The data are not sufficient to warrant definite conclusions with regard to the keeping of cats or dogs in the house. Since cats appear to have a closer association with rats than dogs, and since only seven (17.9%) of 39 persons reported of keeping cats, the relationship between the presence of these animals and the incidence of infection is not great.

The finding that 38.4 percent of the persons raised poultry or lived next to premises where poultry were raised, might be of some significance since rats commonly frequent poultry yards, thus increasing their exposure to human beings.

VI. Susceptibility of Mongooses to Typhus Infection

Mongooses are not only commonly found within various open fields on Oahu but are frequently seen within the urban area of Honolulu. Since these animals commonly feed on rodents, and since they may serve as reservoirs of typhus fever virus, it was thought best to ascertain the susceptibility of these animals to the disease; the two experiments which follow summarize the observations made.

Experiment I.- April 30, 1941.

A grown mongoose, trapped at the Waipio District of Oahu, was obtained through Mr. C. E. Pemberton of the Hawaiian Sugar Planters' Association. The mongoose was inoculated with the Wilmington strain of endemic typhus; a control male guinea pig was also inoculated in order to ascertain the virulence of the strain.

May 10, 1941.

The mongoose was killed; the brain of the animal was removed aseptically and after being emulsified with saline solution was inoculated into four male guinea pigs. Subsequent to inoculation the guinea pigs failed to develop clinical typhus. They were later found susceptible to typhus when inoculated with the Wilmington strain.

Experiment II. - October 17, 1941.

A young mongoose, trapped in the Kaimuki district of Honolulu, was inoculated with the Wilmington strain of endemic typhus.

October 27, 1941.

The mongoose was killed and a saline emulsion of its brain was inoculated into two male guinea pigs. A few days following inoculation the guinea pigs developed clinical typhus (i.e., fever and scrotal swelling). They were subsequently found immune to typhus when inoculated with Wilmington strain of endemic typhus.

The above findings demonstrate the susceptibility of mongooses to typhus. The failure to pass typhus to the mongoose in the first experiment may have been due to an acquired immunity of the animal as a result of a previous natural infection with typhus. It may also mean that mongooses possess a certain amount of natural immunity to the virus. These observations therefore point out that mongooses may serve as a reservoir of murine typhus virus in nature and assist in the spread of this disease from the urban to rural areas.

Plans are underway to determine presence of typhus virus in mongooses trapped in nature. Negative results have thus far been obtained from the inoculation of a guinea pig with emulsion of the brain tissues from four mongooses trapped in the Kaimuki district of Honolulu.

VII. Summary, Discussion and Suggestions for Typhus Control

1. The present study has revealed the following incidence of typhus virus infection among rats trapped in four districts of Honolulu: Kaimuki district, 14 percent; Waikiki, 3 percent; Manoa-Bingham, 9 percent; Downtown, 21 percent. Out of a total of 400 rats examined from these areas, about 11.7 percent have shown infection. It has been suggested that the difference in incidence of infection may be correlated with the abundance of rats in each district and with the opportunity which these animals have to intermingle. There is a likelihood that the incidence of typhus infection among rats in Honolulu if left unchecked would gradually increase. As long as food, shelter, and proper climatic conditions exist, rats and fleas will multiply. Unless rat and flea control are properly established, a typhus epidemic among rats is possible and this in turn increases the risk of human infection.

2. Dogs, cats, and fleas from these animals have been found, under experimental conditions, to be susceptible to typhus infection. However no typhus virus was found in the examination of the brains of 109 cats and 26 dogs from various parts of Honolulu. This finding may indicate that these animals play little part in exposing humans to typhus infection.

3. Local human typhus has been found to be clinically and immunologically similar to the strain of endemic typhus found in local rats. This incriminates rodents as the major source of infection.

4. Of the five species of fleas occurring on rats in Oahu, all except one (X. hawaiiensis) which is very rare, have been shown to be suitable carriers of typhus. The sticktight flea, which is common to rats, dogs, cats, and poultry, has been found for the first time to be susceptible and suitable for typhus transmission. Infection of this flea under natural conditions has been reported.

5. A study on the environmental conditions of 39 local patients recently recovering from typhus fever revealed that 84.6 percent had noticed an abundance of rats, either around their home or place of work. Of these individuals, 17 percent kept cats and/or dogs and 38.4 percent raised poultry or lived next to premises where poultry was kept. Of rats trapped in 17 residences of human typhus cases, murine infection was found in eight or 47.0 percent.

6. Under experimental conditions mongooses were found to be susceptible to typhus infection. It has been suggested that these animals may serve as a reservoir of murine infection and may assist in the spread of the infection in the fields and rural areas.

7. The facts which are thus far known point out that rats are the main host-animals which maintain typhus virus, and fleas are the major source of transfer of the virus among animals and to man. Control measures should therefore be aimed towards the control of rats and fleas. The following suggestions are made:

Rat control may be brought about as below:

1. Rat destruction by (a) trapping, (b) poisoning, (c) gassing, etc.
2. Reduce access of rats to food by:
   (a) Use of proper garbage containers;
   (b) Proper garbage disposal;
   (c) Use of rat guards to prevent rats from climbing trees or entering houses;
   (d) Discourage raising of groups of animals (poultry, hogs, etc.) within city limits and to construct human habitations at a distance from animal quarters.
3. Elimination of rat harborages, by:
   (a) Ratproofing of buildings;
   (b) Discourage construction or maintenance of loose stone walls near human habitations;
   (c) Elimination of, or take proper care of, trees used for nesting by rats;
   (d) Removal of all trash from empty city lots.

Flea control may be brought about by:

1. Control of rats;
2. Control of stray dogs and cats, and preventing access of these animals to basements or under houses;
3. Proper care of pets and poultry so as to eliminate flea infestation.

Additional precautions in typhus control:

1. Do not rub or scratch flea bites.
2. Do not handle rats with bare hand.
3. After handling objects in a place which might have been frequented by rats (such as attic, basement, etc.), do not touch any part of the face unless the hands have been thoroughly washed. Use gloves whenever possible in handling such objects.

C. STUDIES ON LEPTOSPIROSIS

I. Local Importance of Leptospirosis in Man

Leptospirosis or Weil's disease in the Hawaiian Islands is one which has been made known only during recent years, and for this reason its importance is not well known. It may be that this disease is more common than it has been suspected; with a better understanding of this disease locally and the application of better laboratory methods, especially in the use of serological studies, should aid considerably in the better diagnosis of this disease. The 67 human cases which have thus far been reported to the Territorial Board of Health comprise mainly individuals living in rural districts. These cases and localities represented are listed in the following table.

TABLE IV

RECORD OF HUMAN CASES OF LEPTOSPIROSIS
IN THE HAWAIIAN ISLANDS

	Oahu	Hawaii	Maui	Kauai
1936 - 1937	0	28	1	1
1937 - 1938	1	11	14	1
1938 - 1939	0	1	0	0
1939 - 1940	0	3	0	1
1940 - 1941	2	2	0	1
TOTAL	3	45	15	4

II. Murine Leptospirosis

Murine leptospirosis is caused by Leptospira icterohemorrhagiae. The presence of this organism in local rats was first reported by the writer in 1937 (2) following the first observations of human leptospirosis (Weil's disease or infectious jaundice). At that time foci of murine infections were found on the islands of Hawaii and Kauai (4). In the present investigation, which is still in progress, it is desired to determine to what degree and extent murine infection exists locally. Thus far, kidneys of rats secured from four districts of Honolulu have been collected. Of these, 100 kidneys from rats trapped in the Kaimuki district have been examined following staining with the silver impregnation method and sectioning. No infection has been noted in all these tissues studied. The absence of infection might be due to the fact that the rats were trapped around human habitations and empty lots away from swampiness.

In the study of local murine leptospirosis there is a possibility that infection will be found more common among rats associated with fresh water streams, swampy areas, or places where there is considerable rainfall.

One rat trapped along the Waolani stream and another trapped along the Kalihi stream were both found to harbor leptospirae in the kidneys. In addition, out of 12 rats trapped along Nuuanu stream, two were found infected; young mice and guinea pigs inoculated with kidney emulsions of these infected rats developed jaundice. Leptospiral organisms have been isolated in cultures from tissues of these animals. The above findings constitute the first reports of leptospirosis in rats on the island of Oahu.

III. Canine Leptospirosis

Leptospirosis in dogs is caused by Leptospira icterohemorrhagiae and Leptospira canicola, and constitutes a problem of veterinary and public health importance. The former, which is also found in rats, produces jaundice. L. icterohemorrhagiae may be transmitted from dog to dog or rat to dog whereas L. canicola occurs as a result of inter-canine infections. All these infections are brought about through urine contaminations among these animals, and occasionally through the same way to man.

In the present study, leptospiral infections were determined as a result of microscopic agglutination tests made on the sera of 100 dogs from Honolulu and vicinity. Of these sera, 23 were from dogs sent to Dr. Moss' veterinary hospital for various causes and the other 77 sera were obtained from stray dogs in Honolulu. The sera of these animals were tested with fresh formalinized antigens of L. icterohemorrhagiae and L. canicola, and the method adapted was as outlined by Meyer, Stewart-Anderson and Eddie (27). The original strains of these leptospira cultures were secured through the kindness of Dr. K. F. Meyer and Mrs. Stewart-Anderson of the Hooper Foundation.

A detailed report of the examination of the 100 sera of dogs is outlined in the following table.

TABLE V

POSITIVE LEPTOSPIRA AGGLUTINATION TESTS OBTAINED IN THE EXAMINATION OF 100 SERA OF DOGS FROM HONOLULU AND VICINITY

Dog No.	Clinical observations	Titer of Sero-reaction First line: titer for L. icterohemorrhagiae Second line: titer for L. canicola Dilutions 1:10	1:30	1:100	1:300	1:1000	1:3000	1:10000	1:30000	Classification of infection	
1	icteric	2+	2+	2+	1+	o	o	o	o	L. ict.	early clinical(?)
		o	o	o	o	o	o	o	o		
2	general malaise	3+	3+	3+	2+	o	o	o	o	"	"
		o	o	o	o	o	o	o	o		
3	icteric	4+	4+	4+	4+	3+	3+	3+	2+	"	clinical
		4+	4+	4+	3+	2+	o	o	o		
9	bloody urine	3+	3+	2+	1+	o	o	o	o	"	early clinical(?)
		3+	1+	o	o	o	o	o	o		
10	dermatitis	2+	2+	2+	1+	o	o	o	o	"	latent
		o	o	o	o	o	o	o	o		
12	vomiting;leucopenia; dehydrated	4+	4+	4+	3+	3+	1+	o	o	L. can.	clinical
		4+	4+	4+	4+	4+	3+	1+	1+		
13	cough; subnormal temperature	3+	3+	2+	1+	o	o	o	o	L. ict.	latent
		3+	2+	1+	o	o	o	o	o		
16	stomatitis	3+	2+	1+	o	o	o	o	o	L. can.	"
		3+	3+	2+	1+	o	o	o	o		
17	icteric;vomiting	4+	4+	3+	3+	2+	1+	1+	o	L. ict.	clinical
		3+	2+	2+	2+	1+	1+	o	o		
18	normal	2+	1+	o	o	o	o	o	o	L. can.	latent
		2+	2+	2+	1+	o	o	o	o		
19	"	4+	3+	2+	1+	o	o	o	o	L. ict.	"
		1+	1+	o	o	o	o	o	o		
21	vomiting	2+	1+	1+	1+	o	o	o	o	L. can.	"
		3+	3+	2+	2+	1+	o	o	o		
27	malaise;dehydrated;	2+	2+	1+	1+	o	o	o	o	"	clinical
		4+	4+	3+	3+	2+	1+	1+	o		
29	normal	2+	2+	1+	o	o	o	o	o	"	latent
		3+	2+	2+	1+	o	o	o	o		
35	malaise;dehydrated	2+	1+	o	o	o	o	o	o	"	clinical
		4+	4+	4+	4+	3+	3+	3+	2+		
37	normal	3+	2+	1+	o	o	o	o	o	"	latent
		3+	3+	2+	2+	1+	o	o	o		

TABLE V (continued)

Dog No.	Clinical observations	Titer of Sero-reaction First line: titer for L. icterohemorrhagiae Second line: titer for L. canicola Dilutions 1:10	1:30	1:100	1:300	1:1000	1:3000	1:10000	1:30000	Classification of infection	
42	normal	3+ 3+	2+ 1+	2+ o	o o	o o	o o	o o	o o	L. ict.	latent
43	"	3+ 4+	2+ 3+	2+ 2+	1+ 2+	1+ 1+	o o	o o	o o	L. can.	"
46	"	1+ 3+	1+ 3+	o 2+	o 2+	o 1+	o o	o o	o o	"	"
51	"	3+ 2+	3+ 2+	2+ 1+	1+ o	o o	o o	o o	o o	L. ict.	"
53	"	3+ 1+	3+ o	2+ o	1+ o	o o	o o	o o	o o	"	"
55	"	4+ 4+	3+ 4+	2+ 3+	o 1+	o o	o o	o o	o o	L. can.	"
57	"	4+ 4+	3+ 3+	2+ 3+	1+ 1+	o 1+	o o	o o	o o	"	"
59	"	3+ 2+	3+ 2+	2+ 1+	1+ o	o o	o o	o o	o o	L. ict.	"
60	"	3+ 3+	3+ 2+	2+ 1+	1+ o	o o	o o	o o	o o	"	"
61	"	3+ 3+	3+ 2+	2+ 1+	1+ o	o o	o o	o o	o o	"	"
62	malaise; dehydrated; muscular tremor	4+ 4+	4+ 4+	4+ 4+	4+ 4+	3+ 4+	3+ 4+	3+ 4+	2+ 3+	L. can.	clinical
64	normal	2+ 3+	1+ 3+	o 1+	o o	o o	o o	o o	o o	"	latent
67	"	2+ 3+	1+ 3+	1+ 2+	o 1+	o o	o o	o o	o o	"	"
68	"	4+ 2+	3+ 2+	2+ 1+	1+ 1+	1+ o	o o	o o	o o	L. ict.	"
69	"	3+ 2+	3+ 2+	1+ 1+	1+ o	o o	o o	o o	o o	"	"
74	"	4+ 3+	3+ 2+	3+ 1+	2+ o	o o	o o	o o	o o	"	"

TABLE V (continued)

Dog No.	Clinical observations	Titer of Sero-reaction First line: titer for L. icterohemorrhagiae Second line: titer for L. canicola Dilutions 1:10	1:30	1:100	1:300	1:1000	1:3000	1:10000	1:30000	Classification of infection	
78	malaise;dehydrated	4+ 4+	4+ 4+	3+ 4+	3+ 4+	1+ 3+	o 2+	o 2+	o 1+	L. can.	clinical
80	normal	4+ 2+	3+ 2+	3+ 1+	2+ o	1+ o	o o	o o	o o	L. ict.	latent
83	"	3+ 3+	2+ 3+	1+ 2+	o 1+	o o	o o	o o	o o	L. can.	"
84	"	4+ 2+	3+ 2+	2+ 1+	1+ o	o o	o o	o o	o o	L. ict.	"
88	malaise;dehydrated; muscular tremor	4+ 4+	3+ 4+	3+ 4+	3+ 4+	2+ 4+	2+ 3+	2+ 3+	1+ 2+	L. can.	clinical
93	normal	2+ 4+	1+ 3+	1+ 2+	o 1+	o o	o o	o o	o o	"	latent
95	"	3+ 2+	3+ 1+	2+ o	1+ o	o o	o o	o o	o o	L. ict.	"

The data recorded above is summarized in the following table:

TABLE VI

SUMMARY OF POSITIVE AGGLUTINATION TESTS OBTAINED IN THE EXAMINATION OF 100 SERA OF LOCAL DOGS. (From Table V)

Strain of Leptospira	Titer						Total positive
	1:100	1:300	1:1000	1:3000	1:10,000	1:30,000	
L. icterohemorrhagiae	1	15	2	0	1	1	20
L. canicola	1	7	5	0	1	5	19
						GRAND TOTAL	39

The above tables indicate that out of the 100 sera examined, 20 (20 percent) showed agglutinins for L. icterohemorrhagiae and 19 (19 percent) showed agglutinins for L. canicola. Those in which the titer is between 1:100 to 1:1000 are regarded as cases which may have recovered from sub-clinical infection or are possible latent cases, i.e. having acquired the infection sometime previous. Leptospira agglutinins are known to persist in the blood for several years (27). The sera in which the titer is 1:10,000 and over possibly represented active clinical infections. The two listed in the higher titer for L. icterohemorrhagiae were icteric, while the six listed for L. canicola were anicteric and showed general malaise, dehydration and muscular tremor.

The results of the present investigation add to the limited knowledge regarding the geographical distribution and prevalence of canine leptospirosis. This disease is said to be very prevalent in England and continental Europe. It has also been reported from the Netherlands Indies, the Federated Malay States, and Madras (33). In the United States, the North American Veterinarian, v. 22 (12) Dec. 1941, p. 720 lists the infection in 14 states involving Alabama, California, Connecticut, Georgia, Louisiana, Massachusetts, Maryland, Michigan, New Jersey, New York, Ohio, Pennsylvania, Virginia, Wisconsin; also, the District of Columbia and Puerto Rico. Surveys on the prevalence of this disease in the continental United States are few and have been conducted as follows: in northern California (San Francisco and Santa Rosa), New York and Alabama by Meyer and co-workers (27); in southern California (Los Angeles) by Greene (20); in Pennsylvania by Raven and Barnes (30). A summary of these surveys together with the data already presented by us are summarized in the following table.

TABLE VII

REPORTED INCIDENCE OF CANINE LEPTOSPIROSIS IN UNITED STATES

Location	Total dogs examined	Infection with L. can.		Infection with L. ict.		Total infection	
		No.	%	No.	%	No.	%
California							
San Francisco	47	16	34.04	0	0	16	34
Santa Rosa	28	4	14.3	0	0	4	14.3
Los Angeles	368	105	29.0	0	0	105	29.0
New York	111	10	9.0	3	2.7	13	11.7
Alabama	21	0	0	0	0	0	
Pennsylvania	105 ("L. can. 3 times as frequent as L. ict.")					40	38.1
Hawaii							
Honolulu and vicinity	100	19	19	20	20	39	39

From observations made on the continental United States, L. canicola is more common in dogs, than L. icterohemorrhagiae. The survey in Honolulu indicates that the two infections are about equal in distribution.

Meyer and co-workers (27) have in recent years pointed out the public health importance of canine leptospirosis and report 19 known cases of this disease in Europe and two in California. Martmer (25), a Detroit physician, recently reported a case of leptospirosis in a child believed to have contracted it from a dog suffering from the same disease.

The survey conducted in Hawaii has disclosed the important fact that about one-third of the dogs had passed through an attack of leptospirosis, possibly within recent years. From the standpoint of the significance of these findings, one is led to believe that, with the relatively wide distribution of infection in dogs, transmission to man may be anticipated. It must be pointed out that the finding of Leptospira agglutinins in individuals who did not recollect having been jaundiced or passing through a condition diagnosed as influenza, led Meyer and co-workers (27) to conclude: "These and other less carefully investigated mysterious illnesses rather forcibly indicate that human Canicola disease may masquerade in a community in form of atypical, abortive and subclinical infections."

The possibility that latent leptospira infection may be found in the local population instigated a study, now in progress, in which human sera obtained at random are being subjected to a test with different leptospira strains. Up to the present time, out of 50 sera examined, one agglutinated L. icterohemorrhagiae in a dilution of 1:300; this was a Korean male, 65 years of age, a laborer, with no history of having had jaundice.

IV. Summary, Discussion and Suggestions for Leptospirosis Control

1. The studies on the incidence of leptospirosis in rats around Honolulu are not sufficient to warrant definite conclusions. No infection was found in the kidneys of 100 rats trapped around houses and in empty lots of the Kaimuki district of Honolulu. Leptospira infections have been found in rats trapped along Waolani and Kalihi streams; two out of 12 rats trapped along Nuuanu stream were also infected. Emulsion from kidneys of these rats produced jaundice when inoculated in young mice and guinea pigs, and the causative organism has been isolated through laboratory culture methods. This finding is the first evidence of murine leptospirosis on Oahu.

2. A study of canine leptospirosis in 100 dogs from Honolulu and vicinity through the use of microscopic serological agglutination tests revealed that 20 percent showed agglutinins for Leptospira icterohemorrhagiae and 19 percent for L. canicola. It has been pointed out that with such high incidence of canine infection, human infections may be anticipated. Leptospira agglutination tests, aiding in the diagnosis of this disease in humans, should be conducted whenever the infection is suspected.

3. The control of leptospirosis among rats, and the prevention of this infection by man, is largely brought about by rat control. The elimination of stray dogs and cats within the city and the keeping of house dogs within confined quarters as humanely as possible, would probably prevent the spread of this canine infection. Control of human infections may also be brought about by:

   (a) Protection of feet with the use of boots or shoes whenever working in wet and damp areas frequented by rats or dogs. The entrance of rats especially should be prevented in establishments where the floors are kept wet and from basement shower rooms and the like.

   (b) Sick dogs and cats should be given proper veterinary care and should not be fondled; their urine should especially be avoided.

   (c) Swimming or wading in small fresh water streams or ponds frequented by rats should be avoided.

D. STUDIES ON HUMAN TRICHINOSIS

I. Incidence of Infection as Revealed by the Examination of Diaphragms at Autopsy

Within recent years routine autopsy studies in widely separated parts of the continental United States, have revealed a high incidence of infection with trichinae; in a summary of these studies from 1891 to 1937, Sawitz (31) reported that out of 3,322 cases examined, 12.34 per cent showed infection. According to Sawitz, this average percentage, when applied to the living population of the United States, would mean that 16,000,000 people are infected with trichinae.

Human trichinosis was first found in Hawaii in 1936 (2) and up to July 1937 there were 11 cases reported. Since that time and up to July 1941, 30 additional cases have been reported to the Territorial Board of Health. Since the knowledge of trichinae infection locally has been very recent, it was decided to conduct a study to determine its prevalence as indicated by autopsy cases. Up to the present time 133 diaphragms have been examined from autopsies conducted at the Emergency Hospital and Leahi Home. In this examination the writer has been assisted by Mr. Man Hing Au, laboratory technician at Leahi Home. The details of the findings are summarized in Table VIII.

TABLE VIII

TRICHINAE FINDINGS IN HUMAN DIAPHRAGMATIC MUSCLES FROM 133 AUTOPSY CASES

Race represented	Total number examined	Number positive for trichinae	Distribution of infection relative to Sex
Caucasian	23	3	1 female; 2 males
Caucasian-Hawaiian	7	1	1 male
Caucasian-Japanese	1	0	
Chinese	9	1	1 male
Chinese-Hawaiian	2	0	
Chinese-Korean	1	0	
Filipino	17	1	1 male
Fijiian	1	0	
Hawaiian	24	1	1 female
Japanese	23	0	
Korean	7	2	2 males
Puerto-Rican	2	0	
Portuguese	9	0	
Portuguese-German	1	0	
(Unknown)	1	1	?
TOTAL	133	10(7.4%)	7 males 2 females 1 unknown

For the purpose of comparison, the data below shows the incidence of local human trichinosis with that which has been determined in recent years in the continental United States (31).

Locality surveyed	Year	Number of cases examined	Per cent positive
1. Hawaii	1940-1941	133	7.4
2. California	1936	200	24.0
3. District of Columbia	1937	300	13.6
4. Louisiana	1937	200	5.0
5. Massachusetts	1931	58	27.6
6. Minnesota	1934	117	17.0
7. Missouri	1937	1,037	15.3
8. New York	1931	344	17.5

The finding that 7.4 percent trichina infection occurred in the local population reveals that human trichinosis in Hawaii is more common than it has been previously suspected.

II. Summary, Discussion and Suggestion for Trichinosis Control

1. The examination of 133 diaphragms of humans at autopsy has revealed that 7.4 percent harbored trichinae larvae. The incidence of this human disease in the Hawaiian Islands is therefore greater than believed heretofore. Although this incidence is on the average lower than that of most states on the mainland United States, it does constitute, nevertheless, a problem of public health importance.

2. Trichina infection locally is kept going in nature through cannibalism which takes place among rats, mongooses and swine. Domesticated hogs also acquire infection as a result of receiving raw or improperly cooked garbage containing scraps of infected meat. Man acquires the infection as a result of eating improperly cooked pork.

Based on the above facts, the control of human trichinosis rests largely upon the following points:

(a) Rat control;
(b) Proper garbage disposal;
(c) Swine sanitation involving among other things, thorough cooking of garbage before feeding, and ratproofing pens;
(d) Special slaughterhouse cooking, refrigerating or processing of pork products of a kind customarily eaten without being cooked;
(e) Thorough cooking of all pork, domestic or wild, before it is eaten.

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